THE RESPIRATORY EFFECTS OF SMOKE, FUME AND OTHER PARTICULATE INHALATION

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Introduction

The inhalation of smoke, dusts, fumes and other particulate matter are recognised to be a potential cause of lung injury and disease. When considering this matter, it is important to understand that the inhalation of foreign material is a common everyday occurrence. In most cases, this exposure is trivial and leads to minor short-term discomfort or symptoms and does not cause prolonged symptoms or permanent injury or damage to the upper airway or lung. Inhalation of foreign substances may aggravate a pre-existing condition and occasionally cause generalised systemic illness. Inhalation of environmental substances is particularly recognised in the industrial setting where toxic fumes and dusts may be present and, following inhalation, may lead to a variety of lung diseases. Atmospheric pollution has also been recognised to be deleterious to lung health and to lead to the development lung disease. In this regard, there are many examples where smoke, present in the atmosphere for prolonged periods, has caused the worsening of underlying lung disease. For example, the severe smog in the mid-1950s in London led to significant morbidity and mortality in Britain and, in Australia, wood smoke in the Tamar Valley, in Tasmania's north each winter, leads to significant respiratory illness.

Despite improvements in environmental and occupational hygiene and significant advances in ensuring a clean air environment, to protect the community against known risks, events do occur from time to time that overcome protective measures that have been developed and put in place. These include bush and other fires, chemical spills, factory mishaps and others. In order to understand the effects that these situations may have on respiratory and general health, it is important to understand the anatomy and physiology of the respiratory tract.

Anatomical and Physiologic Considerations

The respiratory tract achieves importance in the environmental and occupational health setting because it is exposed to potentially contaminated environmental air. In the normal resting individual, approximately 10,000 litres of air are inspired each day. This figure significantly increases with physical activity. Thus, the potential for airborne substances to cause injury to the respiratory tract is important, particularly in the exercising individual (note that in this context exercise may involve no more than walking). Inhaled substances vary in their potential to cause lung disease. Some have a characteristic odour or rapidly cause significant airway irritation and are quickly recognised. Other substances which are not so easily detected may continue to be inhaled often for prolonged periods, without being recognised (e.g., carbon monoxide). In other situations, exposure is unavoidable, such as those exposed to large environmental fires or who live in communities where winter heating is reliant on burning wood or fossil fuels. Finally, particle size is an important factor that must be considered in their potential development of disease. Particulates are measured in microns and can be any shape, solid or liquid droplets. Those 10 microns or less in size are known as PM10s, those 5 microns or less - PM5s and so on. The large particles are usually trapped in the nose and PM5s are recognised to be able to reach the small airways and alveoli. PM2.5s are small enough to be absorbed into the blood stream through the alveolar-capillary membrane. It is, therefore, incumbent on those individuals and organisations involved to appreciate that early recognition of hazards and risks to health are of paramount importance, that prevention is better than cure and that lung injury may not be reversible.

Thus it follows that the site of disease or injury, caused by inhaled fumes or particulate matter, is determined to a large degree by particle size. Smaller particles will penetrate further than the larger as the airway becomes progressively smaller. In order to better understand the factors involved in the genesis of lung injury and disease secondary to inhaled fumes, aerosols and particulates, it is important to briefly review the anatomical and physiological features of the respiratory tract. This is particularly important, as the lung has significant regional differences in terms of physiology and function.

The respiratory system can be usefully be considered as having four compartments as follows

- the nasopharyngeal compartment - from the nostrils through the nasal passages, throat to the larynx and vocal cords (see Figure 1)

- the tracheobronchial compartment - from the vocal cords to the main airway (trachea), the right and left main bronchi, the progressively smaller airway subdivisions down to the respiratory bronchioles (internal diameter less than 0.5 microns) (see Figure 1 and 2)
- the pulmonary/parenchymal (interstitial) compartment - from the respiratory bronchioles to the alveoli or air-sacs (see Figures 2 and 3)
- the pleural space - the space between the external lining of the lungs and the internal lining of the chest wall (see Figure 1)

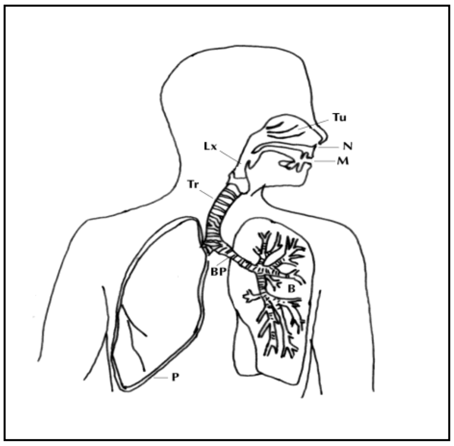

Figure 1: General anatomy

Legend: M – mouth, N – nose, Tu – turbinates, Lx – larynx, Tr – trachea, BP – main bronchus, B – bronchiole, P -

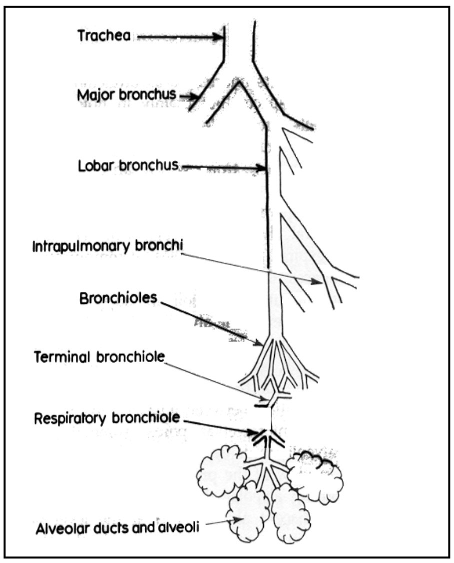

Figure 2: Detailed schematic anatomy from trachea to

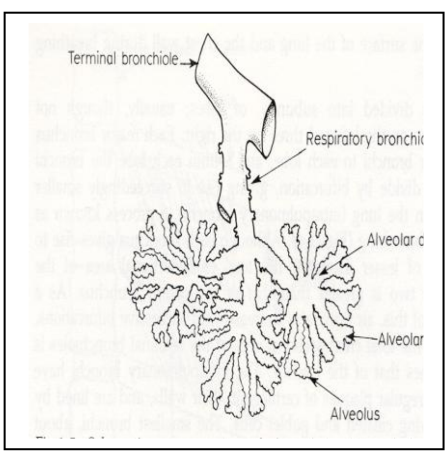

Figure 3: Detailed schematic anatomy of the terminal bronchiole and alveolus

The alveoli present an enormous surface area to the inspired air and act as the interface between the blood and the outside air. They have a rich blood supply, which ensures a direct and unique link between the body and the external environment. In the alveoli, air is separated from the pulmonary capillaries by an extremely thin alveolar-capillary membrane. Its miniscule thickness ensures a rapid exchange of oxygen and carbon dioxide between the blood stream and atmosphere. Whilst the lung is particularly well suited for the uptake of gases, it is also prone to the development of disease or injury as a result of deposition of inspired particulates and absorption of volatile compounds. Substances inhaled as solid or liquid aerosols, gases and vapours all vary in their potential to cause lung disease and may cause irritation or actual physical injury as outlined above.

Materials that can inflict direct injury to lung tissue can lead to respiratory malfunction. This can cause a spectrum of pathological changes including death. Thus, the importance of inhalation as a route of exposure to noxious substances in the environment or workplace cannot be over emphasised. It is not possible, within the limits of this paper to discuss, anymore than briefly, the effects of inhaled foreign material on the lung and the mechanisms by which they cause disease. As outlined above inspired material may take the form of solid aerosols (powders, dusts, smoke), liquid aerosols (mists, fogs, fumes) and gases or vapours, depending on their chemical and physical properties. In the case of smoke, the source is important as the combusted material may contain many substances. For example, the burning of wood, as in a bush fire, may result in different health outcomes when compared to the burning of fossil fuels (coal, oil etc) as the latter may contain amounts of sulphur and organic volatile hydrocarbons.

Broadly speaking, lung diseases can be regarded as those affecting the airways, those affecting the interstitial tissue (the substance of the lung excluding the airways) and some affect both. The following is a list of some examples of these diseases and of their aetiological agents and is illustrative only. Many other factors and agents can affect lung function and structure.

Airway disorders

- Bronchitis (smoke, mineral dusts)
- Bronchiolitis (chlorine, ammonia, volatile organic hydrocarbons)
- Asthma (wood dusts, formaldehyde, isocyanates, grain dust)
- Reactive airways dysfunction syndrome (chlorine, ammonia)

Interstitial disorders

Pneumoconioses (dust disorders)

- Fibrogenic dusts (i.e. scar inducing, e.g. asbestos, silica)
- Non-fibrogenic dusts (coal dust)

Immunological
- Extrinsic allergic alveolitis (foreign proteins, e.g. birds, fungi)
- Lipoid pneumonia (oil, fats)

Alveolar filling disease
- Adult respiratory distress syndrome (phosgene)
- Alveolar proteinosis (silica)
- Lipoid pneumonia (fine oil mists)

Malignancy
- Lung cancer (chemicals in tobacco smoke, asbestos)
- Mesothelioma (asbestos)

Clinical Aspects

Smoke inhalation commonly occurs in the setting of very close contact with a fire, such as in a burning building and, secondly, in the situation were the exposed persons are located remotely from the fire itself. The difference between these two settings is that, in the former, exposed individuals may also be exposed to and inhale hot or super heated air, whereas in the latter the inhaled air is at ambient temperatures and thermal burn injuries, an important contributor to morbidity and mortality, are not experienced. The subject of thermal burn injuries to the airway will not be addressed given that the direction of this report is to consider the subject of smoke inhalation and injury.

The subject of lung disease due to smoke inhalation was brought to the forefront of medical attention in the early 1950s during the London smogs. These were caused by the burning of coal and wood during the winter and led to a large number of excess deaths largely related to respiratory disease. Lung disease was predominated by acute and, subsequently, chronic bronchitis as the inhalation of polluted air continued. We now know that the particulate matter of all sizes contained in smoke is irritative to the respiratory tract. The PM2.5s are the most damaging as these particles penetrate furthest into the airway and reach the bronchioles. Prolonged inhalation causes chronic bronchitis, cough and sputum production. Coal burning may also produce quantities of sulphur dioxide and trioxide and sulphuric acid, all of which are toxic, although the concentrations may vary with the type of coal being burned. Other toxic substances released by the burning of coal include arsenic, mercury, fluorine, cadmium, lead, selenium and zinc. Many of these enter the food chain, notably in fish, and are ingested, thus potentially leading to non-respiratory illness. For example, lead toxicity may affect brain development, causes gastro-intestinal complaints, haemolytic anaemia and fatigue. The elderly and the young are most at risk.

It is recognised that smoke caused by the burning of fossil fuels, wood and any other material (e.g. general waste, crops, forest, bush, grass etc) is a mixture of particles and chemicals. Carbon monoxide, carbon dioxide and other toxic substances, including sulphur dioxide, nitrogen oxides, volatile organic hydrocarbons, metals and other substances may also be released.

The health effects are predominantly on the respiratory system as the upper and lower airway in the portal of entry. Particulates cause non-specific irritation to the airway and lead to the symptoms of cough, sputum production and sometimes a sensation of breathlessness in the short term. More prolonged exposure over several weeks leads to these symptoms becoming more severe and may lead to the development of chronic sinusitis, chronic bronchitis and chronic obstructive airways disease (COPD) in some individuals. The latter are usually permanent or, at least, very long standing. In those persons with pre-existing lung disease, such as COPD or asthma, their condition may be aggravated leading to an increased severity in the symptoms of breathlessness, cough, chest tightness and discomfort. In some cases, the increased severity of the condition will lead to acute respiratory failure necessitating admission to hospital, with or without an Intensive Care Unit admission. Death may occur in some more severe cases. For many persons, cessation of exposure and appropriate medical treatment does not lead to a return to the previous degree of severity of their lung disease, indicating that exposure has led to some further permanent damage to the respiratory system.

Regular and prolonged exposure over many years may lead to cardiovascular disease and lung cancer, in some cases were carcinogens are a constituent of the smoke. For those who recover some loss of lung function is common.

Carbon monoxide is a gas commonly produced by combustion. It is an odourless, colourless gas which is readily inhaled and may cause significant injury and illness if exposure is prolonged or in high concentration. Carbon monoxide has an affinity for haemoglobin that is ten times that of oxygen. It thus displaces oxygen from haemoglobin leading to a reduced oxygen carrying capacity in the blood and thus lowered oxygen supply to the tissues where it is used to generate energy. Symptoms include light-headedness, headache, nausea, impaired cognitive functioning and cardiac dysfunction. These will continue whilst low concentrations of carbon monoxide persist in the atmosphere. In cases where higher concentrations are inhaled unconsciousness may develop and untreated will cause death.

Summary

In summary, smoke inhalation from combusted coal is recognised to cause lung and systemic disease in person with previous good health and to cause worsening of the underlying condition in those with pre-existing lung disease. Cessation of exposure and medical treatment does not necessarily led to a return to the pre-exposure level of health. The literature supports the view that prolonged exposure may lead to an increased mortality rate particularly among those with underlying disease.

Suggested Reading

1. Fogarty PW et al. Long-term effects of smoke inhalation in survivors of the King's Cross underground station fire. Thorax 1991;46:914-918

2. D'Amato G, ST Holgate (editors). The Impact of Air Pollution on respiratory Health. European Respiratory Monograph No 21, 2002, Volume 7

3. Liu D et al. The Effect of Smoke Inhalation on Lung Function and Airway Responsiveness in Wildland Fire Fighters. Am Rev Respir Dis 1992;145:1469-1473

4. Rosenstock L et al. Respiratory mortality among Firefighters. Brit J Ind Med 1990;47:462-465

5. Fogarty PW et al. Long term effects of smoke inhalation in survivors of the King's Cross underground station fire. Thorax 1991;46:914-918

6. Dries DJ et al. Inhalation injury: epidemiology, pathology, treatment strategies. Scan J Trauma Resus Emerg Med 2013;21:31-46

7. Castleden WM et al. The mining and burning of coal: effects on health and the environment. Med J Aust 2011;195:333-33